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EXPERIMENTAL TOXOPLASMOSIS IN BUDGERIGARS (*MELOPSITTACUS UNDULATUS*)

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ABSTRACT: The susceptibility of budgerigars (*Melopsittacus undulatus*) to graded doses of *Toxoplasma gondii* oocysts was studied. Sixteen budgerigars were divided into 4 groups (A–D) of 4 each. Birds in groups A–C were fed 100,000, 1,000, or 100 infective oocysts of the VEG strain of *T. gondii*, respectively. Budgerigars in group D were not fed oocysts and served as controls. All 4 birds in group A died (or were killed) because of acute severe enteritis 5 or 6 days after feeding oocysts (DAFO). Three of the 4 birds in group B were killed (or died) because of toxoplasmosis 9 or 14 DAFO. One budgerigar in group C and the 4 budgerigars in group D remained healthy and were killed 35 or 39 DAFO. *Toxoplasma gondii* was demonstrated in tissues of all budgerigars fed oocysts. The control budgerigars remained clinically normal and showed no evidence of *T. gondii* exposure. These results indicate that, compared to other passerines, budgerigars are relatively resistant to clinical toxoplasmosis.

Toxoplasma gondii infection is subclinical in many avian species, and some species are resistant to clinical toxoplasmosis (Siim et al., 1963; Biancifiori et al., 1986; Hubbard et al., 1986; Parenti et al., 1986; Dubey and Beattie, 1988; Hartley and Dubey, 1991; Dubey, Ruff, Camargo et al., 1993; Dubey, Ruff, Kwok et al., 1993; Literák and Hejlíček, 1993; Dubey et al., 1994, 1995; Literák et al., 1999; Sedlák et al., 2000). Severe toxoplasmosis has been reported in certain species of zoo birds, especially canaries (Vickers et al., 1992; Lindsay et al., 1995; Gibbens et al., 1997; Williams et al., 2001). There is 1 report of toxoplasmosis in 2 naturally infected budgerigars in Switzerland (Galli-Valerio, 1939). The objective of the present report was to study the susceptibility of budgerigars (*Melopsittacus undulatus*) to oral infection with graded doses of *T. gondii* oocysts.

MATERIALS AND METHODS

Infection of budgerigars with *T. gondii* oocysts

The budgerigars were obtained from a local pet store (Dubey and Lindsay, 1998; Dubey, 2000). Oocysts of the VEG strain of *T. gondii* were used, and oocysts had been obtained from the feces of experimentally inoculated cats (Dubey et al., 1996). Oocysts were sporulated in 2% sulfuric acid, and the number of infective oocysts in the inocula was determined by bioassay in mice 4–6 wk before inoculation of bird (Dubey et al., 1996). For this, oocysts were neutralized with 3.3% sodium hydroxide, diluted 10-fold, and aliquots of each dilution were bioassayed in mice. A 10^{-6} dilution of oocysts was infective to 5 of 5 mice. The budgerigars were divided into 4 groups (A–D) of 4 birds each. Birds in groups A–C were fed *T. gondii*, and birds in the fourth group (D) served as uninoculated controls (Table I). The budgerigars were fed aliquots from dilutions 10^{-4}, 10^{-3}, and 10^{-1}; the number of infective oocysts present was estimated to be 100, 1,000, and 100,000 respectively (Table I). The oocysts were deposited directly in the crop by a cannula. The inoculated birds were housed in sterilized wire cages, and their bedding and excreta were collected for 7 days after feeding oocysts (DAFO) and incinerated to kill oocysts that might have passed unexcysted in feces (Dubey and Frenkel, 1973). Budgerigars had access to budgerigar feed ration and sterilized water ad libitum without any anticoccidials.

Examination of budgerigars for *T. gondii* infection

All budgerigars were examined at necropsy. Brain (1/2 brain) and pectoral muscle (~50 g) of budgerigar nos. 142 and 138, killed 35 DAFO, and budgerigar nos. 137 and 140, killed 39 DAFO (Table I), were bioassayed in mice. For this, tissues were homogenized in 0.9% NaCl solution (saline), centrifuged, and suspended in antibiotic saline as described (Dubey, Ruff, Camargo et al., 1993). Tissue homogenate was inoculated subcutaneously into 4 Swiss Webster female outbred 20–25-g mice. Pectoral muscles were digested in acid–pepsin solution (Dubey, 1998), and about half of the digest was bioassayed in 4 mice for each bird.

The mice inoculated with budgerigar tissues were examined for *T. gondii* infection. Imprints of lungs of mice that died were stained with Giemsa and examined microscopically for *T. gondii* stages. The survivors were bled 8 wk later and killed ~4 mo after inoculation with avian tissues. Serum from each mouse was tested for *T. gondii* antibodies at a dilution of 1:50, using the modified agglutination test (MAT), as described by Dubey and Desmonts (1987). A portion of cerebrum from each mouse was examined microscopically for tissue cysts as described (Dubey and Beattie, 1988).

Brain squashes from budgerigars killed 35 or 39 DAFO were examined microscopically for tissue cysts (Dubey and Beattie, 1988), and when tissue cysts were found, the bioassay was not performed.

Histologic and immunohistochemical examinations

Samples of brain, heart, lung, spleen, liver, kidneys, proventriculus, gizzard, intestines, cloaca, tongue, pectoral and leg muscles, and eyes were fixed in buffered neutral 10% formalin. Paraffin-embedded sections were cut at 5 μm and examined after staining with hematoxylin and eosin.

Deparaffinized sections of all tissues were stained with anti–*T. gondii* polyclonal rabbit serum (Lindsay and Dubey, 1989) and anti–BAG-1 rabbit serum (McAllister et al., 1996), as described (Dubey et al., 2001). The polyclonal rabbit serum used in the present study stains all stages of *T. gondii*, whereas anti–BAG-1 serum is specific for bradyzoites (McAllister et al., 1996).

RESULTS

All 4 budgerigars fed the highest dose (100,000 oocysts) became lethargic between 4 and 5 DAFO and died (or had to be killed) 5 or 6 DAFO (Table I). Three of the 4 budgerigars fed 1,000 oocysts died or were killed 9 or 14 DAFO because they were weak (Table I). The fourth bird (no. 142) in this group remained clinically normal and was killed 35 DAFO. All 4 budgerigars fed 100 oocysts and the 4 budgerigars not fed oocysts did not become ill and were killed 35 or 39 DAFO.

Gross lesions were seen in small intestines of budgerigars (group A) necropsied 5–6 DAFO. These lesions were characterized by hyperemia and hemorrhagic to fibrino-necrotic enteritis of small intestine. Microscopic lesions were seen in the small intestine (Fig. 1A, B), liver, cloaca (Fig. 1E), striated muscles, periocular connective tissue (Fig. 2A), bone marrow (Fig. 2B), and the brain (Fig. 2C–E). However, the most severe lesions were in the small intestine.

Intestinal lesions consisted of multifocal areas of mucosal

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TABLE I. Summary of toxoplasmosis in budgerigars fed *Toxoplasma gondii* oocysts.*

Budgerigar no.	Group no.	No. of oocysts fed†	Day died or killed	*T. gondii* in budgerigar tissues		Antibody titer (MAT)
				Immunohistochemical	Lesions‡	
148	A	100,000	D5	I, Lu	I	<25
147		100,000	DK5	I, Lu	I	<25
146		100,000	D5	I, Li	I	ND
145		100,000	DK6	C, I, Li	I	<25
144	B	1,000	DK14	B§, I, M§, Pe	B, C, I, Li, Lu, M, Pe	<25
143		1,000	DK9	B, I, Li, Pr	B, I	100
142		1,000	K35	B		200
141		1,000	D9	I, K, Li, Lu	I	ND
140	C	100	K39	B		200
139		100	K35	B§	B, H	100
138		100	K35	NS‖		25
137		100	K39	NS‖		200
136	D	0	K39	NS	NS	<25
135		0	K39	NS	NS	<25
134		0	K39	NS	NS	<25
133		0	K39	NS	NS	<25

* D = died, K = killed, DK = killed when in extremis, ND = not done, NS = nothing significant. B = brain, C = cloaca, I = intestine, K = kidney, Li = liver, Lu = lung, M = skeletal muscle, Pr = proventriculus, Pe = periocular fat and connective tissue.
† Based on bioassay in mice.
‡ Microscopic.
§ BAG-1 positive.
‖ Bioassay of brain and muscle in mice was positive.

necrosis with extension into the subjacent lamina propria (Fig. 1A) and desquamation of necrotic epithelial cells into the intestinal lumen (Fig. 1B). Numerous tachyzoites were present within the necrotic sites in the lamina propria (Fig. 1C, D). These were present in 4 of 4 birds in group A, 3 of 4 in group B, and none in group C.

Brain lesions consisted of multifocal necrosis and infiltration by mononuclear cells (Fig. 2C, D) with extension to meninges. Tachyzoites were seen within the affected areas. These changes were seen in 2 of 4 birds in group B and in 1 of 4 birds in group C. Tissue cysts were seen in budgerigars killed 14, 35, and 39 DAFO (Fig. 2E).

Focal cholangio-hepatitis, locally extensive nonsuppurative myositis, necrotizing cloacitis, and cellulites of the periocular tissue were seen in budgerigar no. 144, killed 14 DAFO (Fig. 2A).

Toxoplasma gondii was found in histologic sections of tissues of all budgerigars fed 100,000 or 1,000 oocysts and in 2 of the 4 birds that were fed 100 oocysts (Table I). It was seen in intestines of 7 budgerigars, brains and livers of 5, lungs of 4, bone marrow of 1, skeletal muscles, periocular connective tissue, proventriculus, and kidney of 1 (Table I). The organisms that stained positively with anti–BAG-1 serum were seen in leg muscles and in the brain of budgerigar no. 144 and only in the brain of budgerigar no. 139.

Toxoplasma gondii was isolated by bioassay from pectoral muscles of budgerigar nos. 142, 139, 138, and 137 and from brains of budgerigar nos. 142, 140, 138, and 137 (data not shown in Table I).

Antibodies to *T. gondii* were not found in 3 of 4 budgerigars examined 5, 6, or 14 DAFO and in any of the 4 control budgerigars (Table I).

In 2 budgerigars, nos. 133 and 136 (Table I), there was incidental finding of numerous trophozoites of *Giardia* spp. in lumina of intestinal sections.

DISCUSSION

Results of the present study indicate that budgerigars are susceptible to *T. gondii*, and the clinical response is dose-dependent. All budgerigars fed the highest dose (100,000 oocysts) developed severe enteritis, whereas birds fed the lowest dose (100 oocysts) did not develop any obvious clinical signs. Although there are no data on the susceptibility of other avian species to the VEG strain of *T. gondii*, partridges (*Perdix perdix*, *Alectoris graeca*) were found to be highly susceptible to oral infection with oocysts of other strains of *T. gondii* (Dubey et al., 1995; Sedlák et al., 2000).

Sarcocystis falcatula is a coccidian closely related to *T. gondii*. It is infective to a variety of birds, and captive passerine birds in zoos often die of acute sarcocystosis (Box and Smith, 1982; Box et al., 1984; Hillyer et al., 1991; Clubb and Frenkel, 1992). Budgerigars are also highly susceptible to *S. falcatula* infections, and feeding a few sporocysts can be lethal to them (Box and Smith, 1982; Box et al., 1984; Dubey, 2000). Although *S. falcatula* can cause disseminated infection, pneumonia is the predominant lesion with extensive parasitization of intravascular schizonts (Smith et al., 1987). Because zoo environments are often contaminated with *T. gondii* oocysts shed by feral or captive cats in the zoo and with *Sarcocystis* sp. sporocysts shed by feral opossums, budgerigars may acquire both sarcocystosis and toxoplasmosis in the environment. In the present report, the predominant toxoplasmic lesions (enteritis, encephalitis) were distinguished from pulmonic lesions associated with acute sarcocystosis. The location of the organisms is also different in these 2 related protozoan diseases. *Sarcocystis*

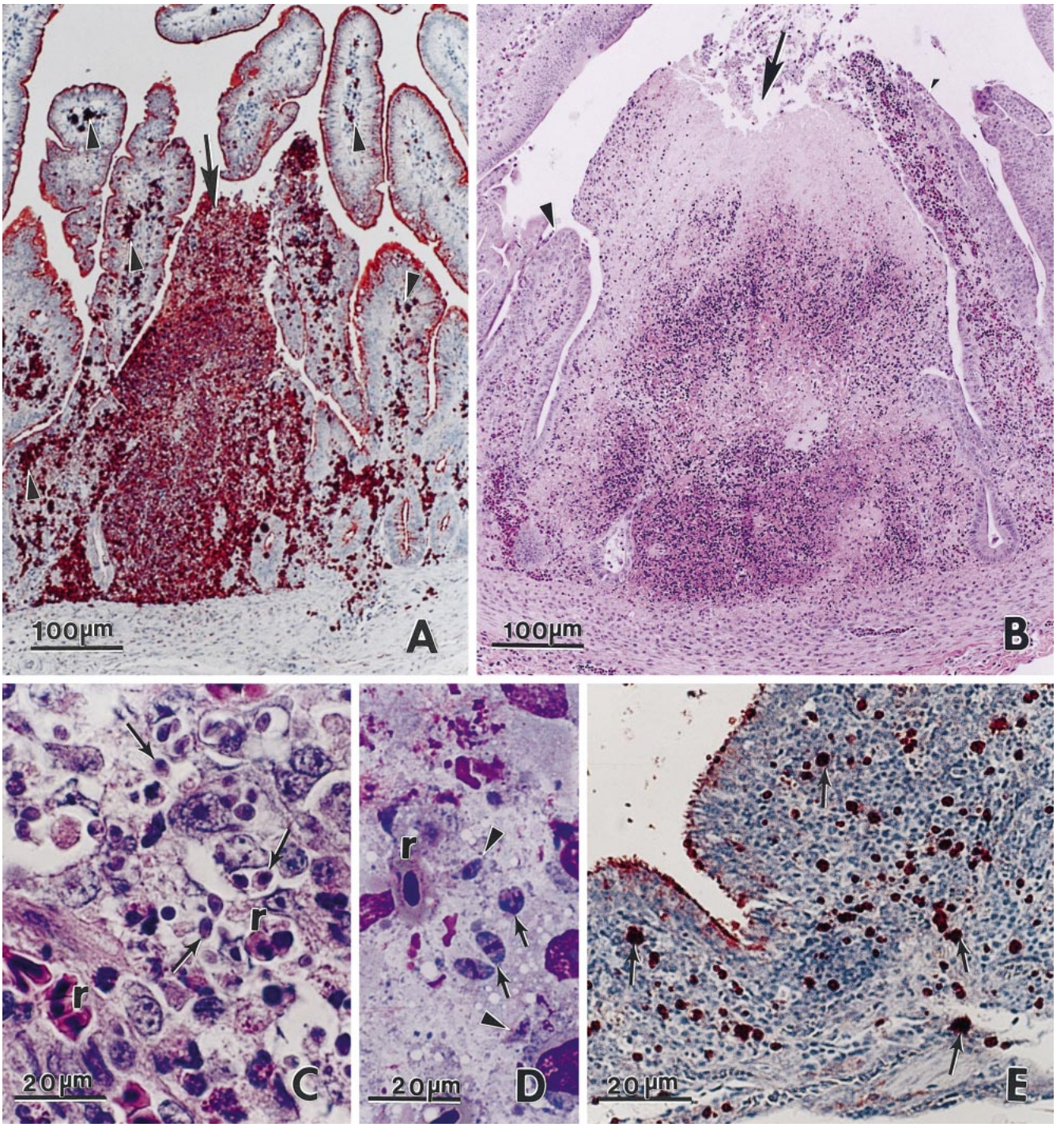

FIGURE 1. Lesions and *Toxoplasma gondii* tachyzoites in small intestine of budgerigars fed oocysts. (**A**) Focal ulceration (arrow) of the mucosa. Numerous tachyzoites (all red spots) are present in the ulcerated area and in the lamina propria of adjacent villi. Budgerigar no. 145, 6 days after feeding oocysts (DAFO). Immunohistochemical stain with polyclonal anti–*T. gondii* serum. (**B**) Focal ulceration of mucosa and necrosis of subadjacent lamina propria. Adjacent villi are intact (arrowheads). Budgerigar no. 143, 9 DAFO, Hematoxylin and eosin (H&E) stain. (**C**) Extensive parasitization by tachyzoites in the lamina propria. Tachyzoites are globular to oval (arrows), stain lighter than host cells, and are difficult to recognize. Compare size with nucleated red blood cells (r). Budgerigar no. 145, H&E stain. (**D**) Smear of intestinal contents. Note dividing tachyzoites (arrows) that are larger than single crescentic tachyzoites (arrowheads). Budgerigar no. 146, 5 DAFO, Giemsa stain. (**E**) Section of cloaca with numerous tachyzoites (arrows) that are present within an area of inflammation. Immunohistochemical stain with anti–*T. gondii* antibodies. Budgerigar no. 141, 9 DAFO.

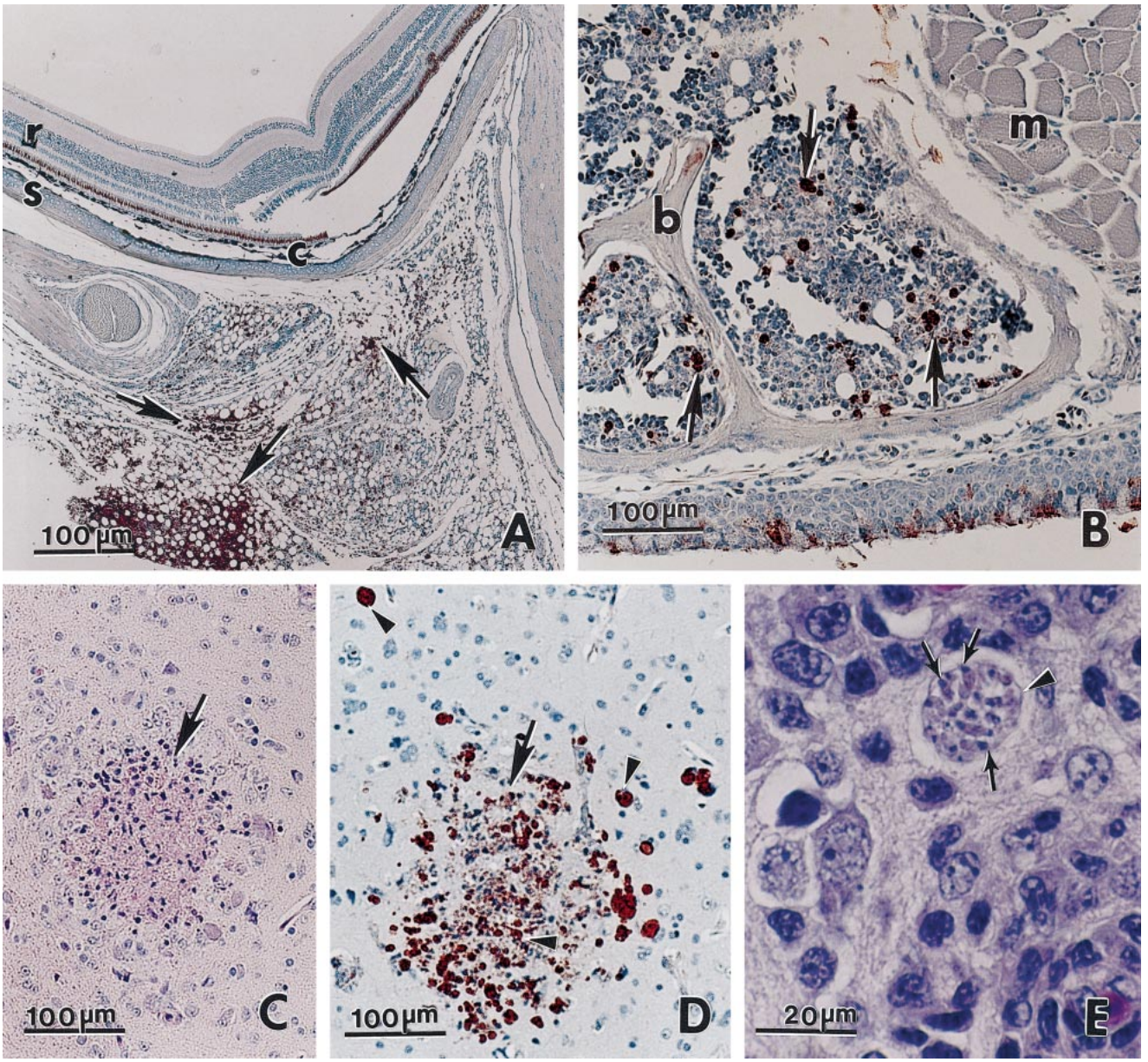

FIGURE 2. Lesions and *Toxoplasma gondii* in tissues of budgerigars fed oocysts. Immunohistochemical stain with anti–*T. gondii* polyclonal serum (A, B, D) and hematoxylin and eosin (H&E) stain (C, E). (**A**) Eye with inflammation and necrosis of periocular tissue. Numerous tachyzoites (arrows, red spots) are present within the area of inflammation. The sclera (s), choroid (c), and retina (r) are normal. Budgerigar no. 144, 14 days after feeding oocysts (DAFO). (**B**) Section of bone (b) with numerous tachyzoites (arrows) in the bone marrow. Note periosteal muscles (m). (**C–D**) Adjacent sections of cerebrum of budgerigar no. 144, 14 DAFO, with focal area of necrosis. Numerous tachyzoites (all red spots, arrowheads) are present in the necrotic area and in the surrounding healthy tissue in the section stained by anti–*T. gondii* antibodies (D) but are not visible in the section stained with H&E (C). (**E**) Granulomatous inflammation with intralesional *T. gondii* tissue cyst (arrowhead) in the cerebrum of budgerigar no. 139, 35 DAFO. Note terminal nuclei of bradyzoites (arrows) enclosed in a thin tissue cyst wall (arrowhead).

falcatula schizonts are found in vascular endothelium, whereas *T. gondii* tachyzoites or tissue cysts are generally located in parenchymal cells of infected organs.

Galli-Valerio (1939) reported *T. gondii* infection in 2 budgeriars (*M. undulatus*) from an aviary with high mortality in Switzerland. Necropsy examination revealed enlarged spleen, congested liver, and no pneumonia. Because there was suspicion of psittacosis, liver homogenates from both birds were inoculated intraperitoneally into mice, which developed toxoplasmosis, and tachyzoites were found in the peritoneal exudate. Rickettsia were not found in tissue smears of mice or budgerigars.

Birds are considered important in the epidemiology of *T. gondii* because they serve as sources of *T. gondii* infection for cats (Wallace, 1973; Ruiz and Frenkel, 1980). However, there is little information available concerning *T. gondii* in passerine birds. In 1 survey, Literák et al. (1997) reported antibodies to *T. gondii* in 28 of 277 (12.3%) sparrows from the Czech Republic. The antibodies were assayed in the indirect fluorescent antibody test (IFAT). Of the 28 seropositive sparrows, IFAT

titers were 1:10 in 21, 1:20 in 6, and 1:40 in 1 sparrow. Thus, most sparrows had titers lower than 1:40, and the significance of these low IFAT titers is unknown. Five of 8 sparrows fed 1 or 10 *T. gondii* oocysts developed low (1:20) IFAT titers, but the parasite was not recovered from their tissues by bioassay (Literák et al., 1997). Ruiz and Frenkel (1980) isolated *T. gondii* from tissues of 16% of 106 sparrows in Costa Rica; all sparrows had no detectable antibodies in the Sabin–Feldman dye test.

In the present study, antibodies to *T. gondii* were found in all 5 budgerigars killed 35 or 39 DAFO. It is of interest that *T. gondii* was not isolated from the brain of the budgerigar no. 138, killed 35 DAI, whereas its pectoral muscle contained *T. gondii*. These results should be taken into account when conducting seroepidemiologic studies. Chances of isolation of *T. gondii* are likely to be higher when both the brain and muscles are sampled together rather than only 1 tissue.

There is also little information on tissue cyst formation in nondomestic avian species infected with *T. gondii*. In the present study, tissue cysts were seen microscopically in the brain by examination of brain squashes of budgerigars killed 5 wk postinfection. Using the reactivity to BAG-1 antibodies, bradyzoites were demonstrable in the brain and skeletal muscle of the budgerigar killed 14 DAFO and in the brain of the bird killed 35 DAFO.

Incidental findings of *Giardia* sp. trophozoites were found in small intestines of 2 of the 4 control budgerigars. Such infections are common in budgerigars (Filippich et al., 1998) and are considered to be nonpathologic incidental findings in these birds. In general, more than 100 budgerigars were used in our laboratory for research on characterization of *S. falcatula*–like organisms (Dubey, 2000) without any problem with these infections or unwanted organisms.

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